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Research Article

Clinical profile of celphos poisoning in central India

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ABSTRACT

Background: Celphos poisoning (trade name of aluminium phosphide) is a large, though under-reported, problem in the Indian subcontinent.
Methods: The study was conducted at the Intensive Care Unit (ICU) of a tertiary care teaching hospital of central India. Data were collected by a retrospective chart review of all patients admitted from February 2010 to February 2014 with a diagnosis of celphos poisoning.
Results: Fifty patients (32 females, 18 males) were registered from the 967 patients of poisoning admitted to the ICU during the same period, of whom 44 (88%) had died (non-survivors) and the remaining 6 (12%) had survived. Forty five cases were of suicidal poisoning, and 5 were of accidental poisoning. Majority [42/50 (84%)] were from rural background. The ingested dose was 7.23 ± 1.28 gram among non-survivors and 3.3 ± 1.9 gram among survivors.
Conclusion: Strict implementation of nationwide pesticide regulation, including restricting the availability of poison, being aware of its toxicity and providing improved medical management in consultation with regional or national poison control centers could further reduce the mortality due to ALP toxicity as there is no antidote available presently.

Keywords: Celphos, Poisoning, Mortality

INTRODUCTION

Celphos poisoning (trade name of aluminium phosphide) is a large, though under-reported, problem in the Indian subcontinent. Aluminium phosphide (ALP), which is readily available as a fumigant for stored cereal grains, is highly toxic, especially when consumed from a freshly opened container.[1,2] Death results from profound shock, myocarditis and multi-organ failure.[3] Aluminium phosphide has a fatal dose of between 0.15 and 0.5 grams (0.0053 and 0.0176 oz).[4] It has been reported to be the most common cause of suicidal death in North India.[5] Despite these large numbers, there has been limited Indian data on the poison characteristic and predictors of mortality in these patients. The purpose of this study was to study the profile of patients presenting with celphos poisoning retrospectively and to identify the factors at admission that might be useful in predicting mortality.

METHODS

The study was conducted at the Intensive Care Unit (ICU) of a tertiary care teaching hospital of central India. Data were collected by a retrospective chart review of all patients admitted from February 2010 to February 2014 with a diagnosis of celphos poisoning. The diagnosis of celphos poisoning was based on alleged history of ingestion of celphos. Forensic reports of gastric aspirates and post-mortem findings were not analyzed. A total of 50 case records of adults aged more than 12 years with celphos poisoning were retrieved. Instances where the patients had presented with an unclear diagnosis of poisoning or where there was consumption of more than

one substance were excluded from the study. The management of celphos poisoning in our hospital is primarily supportive. All patients were admitted into our ICU after initial resuscitation and gastric lavage. A baseline electrocardiogram was recorded and blood samples for biochemical and hematological investigations were sent from the emergency room immediately. Infusion of vasoactive agents and mechanical ventilatory support were instituted where indicated. The baseline demographic data including the amount of celphos ingested, nature of poisoning (homicidal or accidental), and time lag to medical attention from the time of ingestion were collected. Presenting complaints, clinical characteristics (Glasgow coma scale, respiratory distress, blood pressure, pallor), and laboratory characteristics [hemoglobin, total leukocyte count, blood creatinine phosphokinase (CPK) level, arterial blood gas, chest X-ray] at the time of admission were recorded.

The severity of the poisoning was assessed from the extent of organ dysfunction (renal, hepatic, neurological, gastrointestinal, cardiovascular, etc.), the need for mechanical ventilation and the requirement of drugs for vasoactive support.

Statistical analysis was performed using SPSS version 15.0. P <0.05 were considered statistical significant.

RESULTS

Fifty patients (32 females, 18 males) were registered from the 967 patients of poisoning admitted to the ICU during the same period, of whom 44 (88%) had died (non-survivors) and the remaining 6 (12%) had survived. Their median age was 33 ± 12 years. Forty five cases were of suicidal poisoning, and 5 were of accidental poisoning. Majority [42/50 (84%)] were from rural background. The ingested dose was 7.23 ± 1.28 gram among non-survivors and 3.3 ± 1.9 gram among survivors. The time between ingestion and admission to the ICU was 12 ± 1.6 hours among non-survivors and 4 ± 0.9 hours among survivors. Glasgow Coma Scale (GCS) was 14 ± 1. Systolic blood pressure was 82.5 ± 40 mmHg. Epidemiological characteristics of patients are presented in Table 1. Volemic expansion used isotonic saline serum in 90% of patients and colloid in 30% of the patients. 20% of patient had metabolic acidosis and required sodium bicarbonate. 50% patients of non-survivors received magnesium sulphate and 90% patients of survivor group received magnesium sulphate. Twenty-five (50%) patients required vasoactive drugs, and 20 (40%) patients needed mechanical ventilation. The mortality rate was 88% (44 patients). Death occurred after 64 ± 47 hours from the time of admission, corresponding to 72 ± 58 hours from the time of ingestion.

Non-survivors had ingested significantly higher doses of ALP (P <0.0001), had higher time lag between ingestion of celphos to admission to hospital (P <0.0001). Intravenous magnesium sulphate was used in 29 patients. We observed that the use of magnesium sulfate was significantly more among survivors than among non-survivors (P = 0.007).

Table 1: Epidemiological characteristics of patients.

Patient characteristics	Non-survivors	Survivors	P value
Demographic characteristic			
Age in years (Mean ± SD)	35 ± 3.7	32 ± 2.8	Not significant
Gender (%male)	35	38	Not Significant
Poisoning amount of AIP consumed (gm) (Mean ± SD)	7.23 ± 1.28	3.3 ± 1.9	<0.0001
Suicidal poisoning (%)	90%	92%	Not significant
Time lag to ICU transfer	12 ± 1.6 hours	4 ± 0.9 hours	<0.0001
Clinical finding			
GCS at the time of admission (Mean)	14	15	Not significant
Respiratory distress	45%	05%	0.006
Hypotension	72%	40%	0.004
Laboratory findings			
Anemia	10%	06%	Not significant
Leucocytosis	12%	18%	Not significant
Raised CPK	19%	16%	Not significant
Metabolic acidosis	47%	12%	0.003
Abnormal CXR findings	14%	10%	Not significant
Treatment			
Shock at the time of admission	35%	16%	0.002
Need of inotropes	100%	42%	<0.0001
Magnesium sulfate used	50%	90%	0.007
Total stay duration	2 ± 1.1	5 ± 1.9	0.004

DISCUSSION

Several severity factors are described in the literature, such as age, lack of vomiting, severe acidosis, hypotension, hyperglycemia and ingestion of high dose of aluminium phosphide.[6,7] Other factors such as shock, metabolic alkalosis and respiratory failure have been reported in different studies and in our study as well.[8]

ALP is characterized by high mortality, which exceeds 60% and can reach 100%.[9] In our study, mortality rate was 88%. Celphos is formulated as a greenish grey tablet of 3 g, which in the presence of moisture or HCl, releases phosphine. ALP, when ingested, liberates a lot of

phosphine (PH_3) gas in the stomach, which has a very pungent smell. Phosphine gas is rapidly absorbed from the gastric mucosa and, once it gains access to bloodstream, it reaches various tissues and at cellular level inhibits the mitochondrial respiratory chain and hence leads to cell necrosis and death. It has been suggested that phosphine leads to non-competitive inhibition of the cytochrome oxidase of mitochondria, blocking the electron transfer chain and oxidative phosphorylation, producing an energy crisis in the cells.[10]

Gastric lavage is important in the initial stage. The management principles aim to sustain life with appropriate resuscitation measures until phosphine is excreted from the body. If phosphides have been ingested, do not induce emesis. The rationale behind the use of a mixture of soda bicarbonate and coconut oil is guided by the chemical reaction of ALP with moisture and HCl, liberating phosphine gas which rapidly gets absorbed through gastric mucosa. As the poison itself causes a lot of gastric mucosal damage, it exposes a lot of raw area for phosphine absorption. The mechanism by which coconut oil reduces the toxicity of phosphides is unknown but most probably it forms a protective layer around the gastric mucosa, thereby preventing the absorption of phosphine gas. Secondly, it helps in diluting the HCl and again inhibiting the breakdown of phosphide from the pellet. Soda bicarbonate mainly neutralizes the HCl and thus diminishing the catalytic reaction of phosphide with HCl, thereby inhibiting the release of phosphine.[11]

CONCLUSION

Strict implementation of nationwide pesticide regulation, including restricting the availability of poison, being aware of its toxicity and providing improved medical management in consultation with regional or national poison control centers could further reduce the mortality due to ALP toxicity as there is no antidote available presently.

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